CASE REPORTS

Refer to: Rhame FS, Werner SB: Vibrio parahaemolyticus gastroenteritis from eating conchs. West J Med 121:66-68, Jul 1974

Vibrio Parahaemolyticus Gastroenteritis from Eating Conchs

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VIBRIO PARAHAEMOLYTICUS was first recognized as a cause of human gastroenteritis in 1950 in Japan.[1] It is now recognized as one of Japan's most common causes of foodborne illness.[2-4] In the warm summer months it accounts for up to 50 percent of cases. The organism is widely distributed in the coastal waters of the world, including many areas of the United States. It has been found in a variety of marine fish, shellfish, mud, sediment and water samples obtained primarily from offshore locations. Soft tissue infections[5] caused by V. parahaemolyticus have occurred, but most disease and all outbreaks have been limited to gastroenteritis caused by contaminated seafood. In 1971 it was confirmed as a cause of foodborne illness in the United States.[6] In the 13 outbreaks reported from this country to date, cases have been traced to contaminated oysters, crab, shrimp, and lobster.[7] The present case may have originated from conch meat taken from waters near the Bahamas; it is the first time that this disease has been confirmed in California.

Report of a Case

A 38-year-old black housewife was admitted to hospital February 20, 1973, because of watery diarrhea and lower abdominal cramps. Symptoms had begun 23 hours after she had eaten from conchs which had originated in the Bahamas and had been bought at a Miami fish market. The conchs had been packed in ice, air freighted to California and eaten the day they were received. The patient's lower abdominal pain was sudden in onset and was followed shortly by profuse watery diarrhea and vomiting. She denied chills, headache and myalgias. In the 48 hours before the onset of symptoms, she had eaten only dry or canned foods except for Polish link sausage.

On admission, she was in minimal distress. Temperature was 36.6° C, pulse 84 and regular, blood pressure 140/100 mm of mercury. The abdomen was moderately tender diffusely, and bowel sounds were intermittently active, with no pathological sounds. The remainder of the physical examination was unremarkable. Laboratory data included leukocyte count of 25,700 cells per cu mm on admission, with 82 percent polymorphonuclear cells, 7 percent banded forms, 6 percent lymphocytes, 3 percent monocytes, and 2 percent eosinophiles. Serum sodium was 139, carbon dioxide 27, potassium 3.9, and chloride 105 mEq per liter. Serum urea nitrogen, hemoglobin content, and results of urinalysis, examination of the chest and an electrocardiogram were within normal limits. Two stools were negative for occult blood. An abdominal x-ray film showed diminished bowel gas. On sigmoidoscopy 18 hours after the onset of symptoms the lumen could be viewed only to a distance of 15 mm but in that portion there were no abnormalities. Stool cultures on admission and 18 hours later, as well as culture of a sigmoid colon swab, were positive in heavy growth for V. parahaemolyticus on thiosulfate-citrate-bile salts-sucrose (TCBS) agar[8] but negative for enteric pathogens on routine media. The isolate was confirmed at the California Microbial Diseases Laboratory, and it was found to be type 04:K12, Kanagawa-positive at the Center for Disease Control, Atlanta. On Kirby-Bauer antibiotic susceptibility testing,[9] the tetracycline disc produced a zone of inhibition measuring 22 mm in diameter. Growth on Mueller-Hinton agar was poor.

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Submitted October 26, 1973.

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The patient was treated with intravenous fluids and perchlorpromazine but no antibiotics. Nausea and vomiting ceased within several hours of admission. Diarrhea diminished in frequency and severity, and she was discharged two days after admission. She remained afebrile throughout the illness.

She returned February 26 with recurrence of diarrhea, lower abdominal pain and vomiting. The physical examination was unremarkable except for right lower quadrant tenderness on deep palpation. Leukocytes numbered 29,100 per cu mm with 64 percent polymorphonuclear cells, 26 percent banded forms, 2 percent lymphocytes and 8 percent monocytes. Serum electrolytes, total bilirubin, alkaline phosphatase, lactic acid dehydrogenase and glutamic transaminase were within normal limits. A rectal swab yielded V. parahaemolyticus in small numbers. Tetracycline was given by mouth, 500 mg four times a day (for seven days), and the symptoms resolved two days later.

The patient's husband and two of her children also had eaten small portions of conch on February 19. One of the daughters had diarrhea on February 25. Cultures of rectal swabs from this daughter and the husband on February 26 were negative for enteric pathogens on routine media and TCBS agar. A specimen of the implicated conch which had been refrigerated for only three days was negative for V. parahaemolyticus on March 6.

Discussion

Vibrio parahaemolyticus is an enteropathogenic, facultatively anaerobic, Gram-negative rod which prefers alkaline conditions and a salt concentration of 2 to 4 percent. Unless this organism is specifically sought, it may not be discovered on routine culture for enteric bacterial pathogens. It grows with more difficulty than V. cholerae on MacConkeys medium. Neither of these organisms grows on Salmonella-Shigella agar or EMB agar. Both grow well on TCBS agar, which has a high salt concentration and is alkaline (pH 8.6). In the present case, TCBS agar was used because of the history of recent consumption of an uncooked seafood product. Many cases of gastroenteritis due to V. parahaemolyticus undoubtedly go unrecognized because of failure to culture stool specimens on appropriate media. Stable dehydrated TCBS agar is commercially available. Bacteriology laboratories may stock it for use when the patient's food history is appropriate. Nonselective media may be used if no TCBS agar is available. Since the clinical symptoms are similar to those of salmonellosis and shigellosis, V. parahaemolyticus must be considered in the differential diagnosis of gastroenteritis,[10] particularly when seafood is identified as the cause of a foodborne outbreak.

The relative importance of enterotoxin and invasion in the pathogenesis of V. parahaemolyticus gastroenteritis has not been established. Isolates from diarrheic stool almost always demonstrate the Kanagawa phenomenon while isolates from the environment usually do not.[11] The heat-stable hemolysin responsible for the Kanagawa phenomenon lyses human erythrocytes (Wagutsuma's agar) but not equine erythrocytes.[12] Cell-free culture filtrates (containing the hemolysin) are reported to have no effect on ligated rabbit ileal loops, while live cell suspensions cause reaction.[11] Yahagi has reported that V. parahaemolyticus penetrates epithelial cells and the lamina propria of the ligated rabbit gut,[13] as do shigellae, also favoring the importance of invasion.

Illness from V. parahaemolyticus is generally of sudden onset. The incubation period is usually about 12 hours, but it may vary, from 2 hours to 48 hours.[5] Illness is generally brief and self-limited; death from this disease has only recently been reported in the United States.[14] Antibiotic therapy of cholera shortens the duration of diarrhea and the excretion of V. cholerae.[15] However, antibiotic therapy of V. parahaemolyticus gastroenteritis is not of proved value. The relationship of tetracycline therapy and the remission of the symptoms in the present case cannot be established. Little can be concluded from the susceptibility test reported here, as disc testing for V. parahaemolyticus has not yet been standardized.

Failure to isolate V. parahaemolyticus from the conch meat was disturbing; this could have resulted from overgrowth of other organisms or die-off of V. parahaemolyticus during the six days between the date of ingestion and the date of culture. Negative results on culture of rectal swab specimens taken from other members of the patient's family may have been the result of the long interval (seven days) between ingestion and culture. Indeed, as with most gastroenteritis, the opportunity for isolating the organism diminishes quite rapidly—an argument for early cultures.

Another possibility is that the family members suffered a coincidental and unrelated illness.

Little information is available on the carrier state of V. parahaemolyticus and its public health significance. There has been no evidence of secondary transmission in any of the 13 United States outbreaks. No long-term carriers were identified in the large outbreaks reported from Maryland.[16] Eight-tenths of 1 percent of healthy food workers and 7 percent of healthy sushi cooks in Japan were found to carry the organism during summer months, but the strains found were Kanagawa-negative.[2] Foodborne outbreaks of V. parahaemolyticus are preventable by appropriate cooking and refrigeration practices, and by avoiding contamination of cooked products by raw ones or surfaces and implements that have had contact with raw fish products. Control of the problem in Japan will be difficult, considering the popularity of raw and partially cooked seafood in the Japanese diet.

The available data on *in vitro* antibiotic susceptibility testing are confusing. Studies of disc tests have either used very high antibiotic contents,[17] failed to report zone sizes,[18] or used nonstandard agar medium.[19] These studies have been interpreted as showing sensitivity to tetracycline.[20] However, 13 strains of V. parahaemolyticus tested by the "gutter plate method" showed complete inhibition to tetracycline at 10 μgm per ml with partial inhibition starting at 5 μgm per ml,[18] and five strains tested by a tube dilution technique showed inhibition by oxytetracycline at 5 μgm per ml but not 2.5 μgm per ml. The break-point for tetracycline sensitivity is generally considered to be $\leq$ 4 μgm per ml.[21] It has been suggested that satisfactory disc testing may be performed with trypticase soy broth and Mueller-Hinton agar, both supplemented with 3 percent sodium chloride.[12]

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